the screw-on cap were embedded in the cranial cavity (Fig. 3). On presentation the child was obtunded and had a right third nerve palsy and moderate left-sided weakness.

When he was seen in the neurosurgical department the level of consciousness had improved, but he still had the same neurological deficit. There was an oval, right temporal skin defect. The anterior portion of this flap was driven into the skull, together with the bottle fragment. Brain was exposed posteriorly and was pulsating.

Carotid angiography was carried out. The right middle cerebral vessels were displaced medially and the anterior cerebral artery was shifted across the midline (Fig. 4). In theatre the wound was extended and a craniectomy carried out. The neck of the bottle and the indriven flap of skin were removed. Bleeding from the veins of the Sylvian fissure was controlled. In-driven pieces of bone and hair were removed from the pituitary and carotid areas. The third nerve was intact on exposing the area, but there was some contusion of the frontal lobe. The wound was drained for 3 days and heavy antibiotic cover (intravenous penicillin 2 mU every 2 hours, intravenous sulphadiazine 1 g every 6 hours, and intramuscular streptomycin 0,25 g every 12 hours) and tetanus toxoid were given.

The child made an excellent recovery and was returned to the referring hospital on the 34th day with a residual, very minimal, right hemiparesis and a third nerve lesion.

DISCUSSION

Many unusual objects have been responsible for ocular and cerebral penetrating injuries.[1-3] However, the two cases presented were caused by very unusual objects under extremely unusual circumstances. They are a reflection of the bizarre circumstances often faced by neurosurgeons in a busy general hospital.

Both cases demonstrate the influence of the bony cranial structures on foreign bodies entering the temporal area or the orbit, and the potential vulnerability of the basal major vessels.[2,4,5]

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Herpesvirus hominis Oesophagitis and Oesophageal Stricture

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SUMMARY

The literature on herpetic involvement of the oesophagus is reviewed and a case is described in which the presumptive clinical diagnosis of primary *Herpesvirus hominis* stomatitis and oesophagitis and subsequently oesophageal strictures was made. The differential diagnosis of an oesophageal lesion and its treatment are discussed.

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Most of the cases of herpetic oesophagitis reported in the literature occurred in association with debilitating disease,[1] a thoracic malignant tumour under treatment with radiotherapy and/or surgery, chemotherapy,[2] immunosuppression[3] or malnutrition.[4] Cases have also been described[5,6] in apparently immunocompetent patients. In none of these reports was the condition complicated by stricture formation.

The purpose of this paper is to report the case of a child in whom multiple oesophageal strictures were diagnosed a few months after the presumptive diagnosis of a primary *Herpesvirus hominis* (HVH) stomatitis and oesophagitis had been made. In the interval two further episodes of stomatitis of undetermined aetiology had occurred.

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CASE REPORT

A 3-year-old Coloured girl was referred to this hospital in July 1978 for investigation of dysphagia and vomiting of 2 weeks' duration. This had progressed to a stage at which she could not even swallow her own saliva. At this time she presented with severe dehydration and weight loss.

The previous history was as follows: since the age of 3 months she had had epileptic seizures, for which she had been treated with phenobarbitone. Three siblings had died of 'epileptic convulsions' and her brother aged 5 years was also an epileptic.

On 8 March 1978 she had been admitted to another hospital with ulcerative stomatitis involving the tongue, buccal mucosa, lips and face, and with severe odynophagia (pain on swallowing). The clinical picture was typical of HVH stomatitis. On the following day she developed measles, and a nasogastric tube was passed because of severe pain on swallowing. She recovered well from the measles and stomatitis and was discharged on 17 March.

After discharge she had two more attacks of ulcerative stomatitis. The nature of the first recurrence in April was apparently infectious, but laboratory investigations were not carried out. She was treated at home by the private practitioner with dequalinium, povidone iodine mouthwash, and gentian violet applied locally to the ulcerated mucosa. The second recurrence on the 13 June necessitated admission to a private hospital for parenteral feeding and nasogastric intubation, since she was unable to swallow. The aetiology was apparently infectious; it was possibly a herpangina such as may be caused by Coxsackie A virus infection. Again virological investigations were not carried out. She was discharged after a week, but increasing dysphagia soon led to the vomiting of small volumes of food at each feed.

There was no history of injury of the oesophagus due to the swallowing of corrosives or hot food. The parents are intelligent and the child had always been looked after by the mother herself. A history of oesophageal reflux could not be elicited, and there was no history compatible with epidermolysis bullosa.

Physical examination on admission in July 1978 revealed a very irritable and severely dehydrated but otherwise normal-looking child. The ulcerative stomatitis had healed without scarring. Mobility of the tongue and the appearance of the oral cavity and teeth were normal. There was no evidence of bullae or scarring of the skin of the trunk or extremities, and no webbing of the fingers or contractures. The nails were normal. Laboratory investigations showed the haemoglobin concentration to be 9,1 g/dl; the total serum protein value was 67,0 g/l and that of albumin 32,2 g/l. There was a total white cell count of 10,7 $\times$ 10^9/l, and a differential count with mainly polymorphonuclears and 35% lymphocytes. A week after admission the values for IgG were 9,61 g/l, for IgA 0,76 g/l and for IgM 1,17 g/l.

Previous HVH infection was confirmed by the finding of a serum neutralizing antibody titre of 80, but unfortunately no virological investigations had been carried out during the acute episodes of illness.

On radiological examination the heart and lungs were normal. The barium swallow revealed a normal cervical oesophagus, but from the level of D4 to the diaphragm the thoracic oesophagus showed a continuous irregular narrowing, which did not relax with the intravenous administration of hyoscine butylbromide and was caused by stricture formation (Fig. 1). The gastro-oesophageal junction functioned normally without evidence of spasm or gastro-oesophageal reflux, and a normal amount of air was present in the gastric fornix.

After intravenous rehydration, oesophagoscopy was performed under general anaesthesia; this revealed severe fibrotic narrowing without any evidence of active ulceration or candidiasis. Since it was felt that repeated dilatations at short intervals would be necessary, a gastrostomy for feeding was performed at the same time.

The differential diagnosis of the oesophageal lesion included the following:

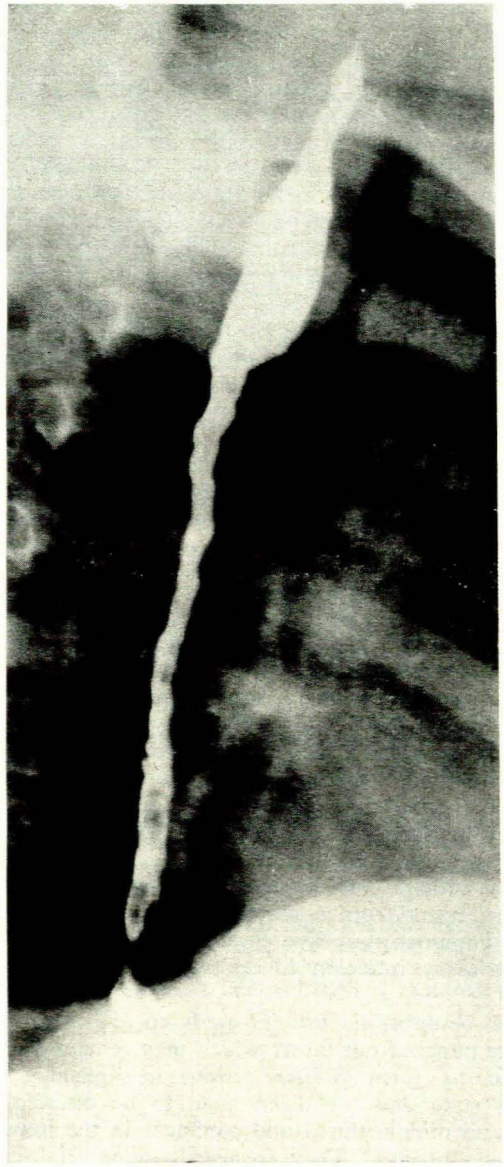

Fig. 1. Long stricture of thoracic oesophagus presumably due to HVH oesophagitis.

1. Corrosive oesophageal stricture seemed unlikely because of the negative history and because the patient had been attended by a medical practitioner during the episodes of stomatitis.

2. Peptic oesophagitis due to hiatus hernia and reflux of gastric contents usually affects the lower part of the oesophagus only. There was no history or radiological evidence of gastro-oesophageal reflux, and the condition could be excluded.

3. Candidiasis usually produces a less severe degree of narrowing, but may complicate HVH infection.[7] In this case there was no evidence of candidiasis at oesophagoscopy, but its earlier occurrence cannot be excluded.

4. Epidermolysis bullosa, a genetic condition, may produce a similar oesophageal stricture in children and adults. In all the described cases, however, there was a history of 'fragile skin' and epidermal bullae followed by scarring, contractures and loss of teeth and nails.[8-12] Our patient did not manifest any of these features.

5. Primary HVH oesophagitis followed by oesophageal strictures. The initial infection can be accepted on clinical grounds as a typical primary HVH stomatitis, and the concomitant odynophagia which necessitated intubation seems to justify the diagnosis of an associated HVH oesophagitis. Cell (thymus-derived lymphocyte)-mediated immunity is important for recovery from HVH infection. Measles is well known to depress the cell-mediated immunity, and some other viral or bacterial infection may have occurred. The nasogastric tube may have induced gastric reflux, aggravating the oesophagitis.

The role of the subsequent two episodes of stomatitis is open to speculation. It is very unlikely that these were due to HVH infection. The nature of the first recurrence is not clear, but it was treated by the medical practitioner at home as an infection. The second recurrence required hospitalization and intubation and on clinical grounds it appeared to be an infection, possibly a herpangina, which is usually caused by a Coxsackie A virus.

DISCUSSION

HVH is a DNA-containing virus with an enveloped icosahedral capsid, known to cause a wide variety of lesions in virtually all the tissues of the body. It seems to be less generally recognized as causing ulcerative oesophagitis, which is usually first diagnosed at autopsy.[5] However, in reported autopsy series a herpetic aetiology was established in from less than 2% to as many as 25% of patients with oesophageal ulceration.

Many authors consider that most cases of HVH oesophagitis[13] result from a reactivation of a latent infection due to immunosuppressive circumstances, but it can occur as a primary infection in apparently immunocompetent hosts.[5,6]

HVH oesophagitis usually leads to the formation of multiple punched-out ulcers which may remain discrete or coalesce to form diffuse erosive oesophagitis.[2] Some authors state that the ulcers tend to be discrete in the upper and middle thirds and confluent in the lower third of the oesophagus.[14] They are usually superficial, but may penetrate deeply into the wall,[3,4,14] and the patient may present with significant haemorrhage.[6]

They have been described at all sites in the oesophagus, but most frequently occur in the lower 3 - 4 cm or at the level of the thyroid cartilage. At the latter site it is also common to find nonspecific ulceration due to nasogastric intubation.

Superinfection with fungi such as candida,[3,7] aspergillus,[2] and cryptococcus[2] may both aggravate and obscure a primary HVH infection, but fungi may sometimes be the primary cause of ulceration.

In addition to the predisposing diseases and therapy referred to in the introduction, some authors also believe that nasogastric intubation is of prime importance.[2,14] We suspect that, apart from the pressure trauma of a nasogastric tube *in situ*, the tube could also lead to aggravation of oesophageal ulceration due to reflux of gastric contents.

Diagnostic virological studies were not carried out during the acute episodes in this patient, but previous HVH infection was confirmed by the serum neutralizing antibody titre of 80.

A definitive diagnosis is not possible in this patient because of the inherent difficulties in attempting to make a retrospective diagnosis and because of inevitable reservations about completely excluding the ingestion of corrosives in a 3-year-old child. Could the combination of primary HVH infection with the immunodepressant effect of concomitant measles, and the nasogastric tube with possible gastric reflux as local traumatizing factors, be a possible explanation for the pathogenesis of the oesophageal strictures in this patient? Could the two subsequent episodes of stomatitis within a period of several months also have played an aetiological role?

If these are possible explanations and if adequate intravenous feeding is not practical in a child with severe infectious oesophagitis and odynophagia, the insertion of a gastrostomy feeding tube should be considered as an alternative to a nasogastric tube.

Since her admission to hospital the patient has gained 3 kg in weight and attends hospital as an outpatient for follow-up dilatation of the oesophagus. She swallows without difficulty, and the narrow segment admits a 29 EG gum elastic bougie with ease.

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